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3: Bochner BS, Bickel CA, Taylor ML, MacGlashan DW Jr, Gray PW, Raport CJ, Godiska R. Related Articles, Links

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 NEWS 30  Oct 24   Nutraceuticals International (NUTRACEUT) now available on
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         29160 ALLERGY
          2264 ALLERGIES
         29817 ALLERGY
                 (ALLERGY OR ALLERGIES)
           339 CCR4
L1          20 ALLERGY AND CCR4

=> allergy and MDC
         29160 ALLERGY
          2264 ALLERGIES
         29817 ALLERGY
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                (ALLERGY OR ALLERGIES)
           587 MDC
            42 MDCS
           610 MDC
                (MDC OR MDCS)
L2          12 ALLERGY AND MDC

=> treatment and L1
       1721072 TREATMENT
        160234 TREATMENTS
       1809421 TREATMENT
                (TREATMENT OR TREATMENTS)
L3          6 TREATMENT AND L1

=> treatment and L2
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L4   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:526192  CAPLUS
DOCUMENT NUMBER:          135:117975
TITLE:                    Regulatory sequence for dendritic cell-specific
                          expression from human fascin genes and their use
INVENTOR(S):              Reske-Kunz, Angelika; Ross, Xiaolan; Ross, Ralf;
Bros,
                          Matthias
PATENT ASSIGNEE(S):       Germany
SOURCE:                   PCT Int. Appl., 117 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 German
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
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     WO 2001051631      A2   20010719       WO 2001-EP362    20010112
     WO 2001051631      A3   20020418
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
             HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
             LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
             SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
             YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
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             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     DE 10001169        A1   20010726       DE 2000-10001169 20000113
     EP 1250430         A2   20021023       EP 2001-903644   20010112
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.:                  DE 2000-10001169 A  20000113
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DE 2000-10010188 A 20000302
WO 2001-EP362 W 20010112

AB The invention relates to regulatory sequences which impart a specific expression in dendritic cells. The regulatory sequences are isolated from the human fascin gene and also comprise, for example, promoter sequences. The invention also relates to recombinant nucleic acid mols. and vectors, which contain the regulatory sequences, and to preferred embodiments of the recombinant nucleic acid mols. and vectors, which code the antigens or immunoregulatory proteins. The invention addnl. relates to host cells, which contain the recombinant nucleic acid mols. or vectors, and to methods for the prodn. thereof. Addnl. embodiments relate to in-vitro methods for stimulating T cells and for producing T cell-stimulating dendritic cells, and to their formulation as medicaments. Addnl. medicaments are described which essentially relate to DNA vaccines and to gene-therapeutic medicaments, for example, for the immunization against and for the **treatment** of infectious diseases, tumors, **allergies**, Creutzfeldt-Jakob plaques or Alzheimer plaques. Addnl. inventive medicaments can be used for the targeted modulation of immune responses that is imparted by dendritic cells, for example, for treating autoimmune diseases or transplant rejection. Finally, the invention relates to different uses of the regulatory sequences. Cloning of the gene and anal. of the 5'-flanking region using a luciferase reporter gene are described.

L4 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:223049 CAPLUS
DOCUMENT NUMBER: 130:251233
TITLE: Macrophage-derived chemokine (**MDC**), **MDC** analogs, **MDC** inhibitor substances,and their therapeutic applications
INVENTOR(S): Gray, Patrick W.; Chantry, David H.; Deeley, Michael C.; Raport, Carol J.; Godiska, Ronald
PATENT ASSIGNEE(S): Icos Corporation, USA
SOURCE: PCT Int. Appl., 159 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9915666	A2	19990401	WO 1998-US20270	19980928
WO 9915666	A3	19990916		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, US, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CN 1163635	A	19971029	CN 1996-190875	19960607
US 5932703	A	19990803	US 1996-660542	19960607
CA 2302806	AA	19990401	CA 1998-2302806	19980928
AU 9897778	A1	19990412	AU 1998-97778	19980928
EP 1017818	A2	20000712	EP 1998-951961	19980928

R: AT, BE, CH, DE, ES, FR, GB, IT, LI, SE, IE

PRIORITY APPLN. INFO.: US 1995-479620 A2 19950607
US 1995-558658 A2 19951116
US 1996-660542 A2 19960607
US 1997-939107 A2 19970926
US 1998-67447 A2 19980428
WO 1998-US20270 W 19980928

AB The present invention provides purified and isolated polynucleotide sequences encoding a novel macrophage-derived C-C chemokine designated "Macrophage Derived Chemokine" (**MDC**), and polypeptide fragments and analogs thereof. **MDC** cDNA sequences and their deduced amino acid sequences are provided from human, mouse, rat, and macaque. Also provided are materials and methods for the recombinant or synthetic prodn.
of the chemokine, fragments, and analogs; and purified and isolated chemokine protein, and polypeptide fragments and analogs thereof. Also provided are antibodies reactive with the chemokine and methods of making and using all of the foregoing. Also provided are assays for identifying modulators of **MDC** chemokine activity. **MDC** possesses antiproliferative activity against HIV-1 virus, stimulates fibroblast proliferation, inhibits tumor growth, induces chemotaxis of TH2 helper T cells, and modulates platelet aggregation, and is shown to be a high-affinity ligand for CCR4.

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L3 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:832576 CAPLUS
TITLE: **Treatment** of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 764 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085309	A2	20021031	WO 2002-US13143	20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-286036P P 20010424

AB This patent relates to a compn. comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addn., they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the **treatment** of bronchoconstriction and/or inflammation. These agents and the compn. and formulations provided are suitable for the **treatment** of respiratory tract, pulmonary and malignant diseases assocd. with bronchoconstriction, respiratory tract inflammation and **allergies**, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as **allergies**, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis, RDA, COPD, and cancers, among others. The present agents and compn. may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L3 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:832575 CAPLUS
TITLE: **Treatment** of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 872 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085308	A2	20021031	WO 2002-US13135	20020423

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WO 2002085308 A2 20021031 WO 2002-XA13135 20020423

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WO 2002085308 A2 20021031 WO 2002-XB13135 20020423

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PRIORITY APPLN. INFO.: US 2001-286137P P 20010424
WO 2002-US13135 A 20020423

AB This patent relates to a compn. comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addn., they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the **treatment** of bronchoconstriction and/or inflammation. **Treatment** with antisense oligonucleotides in combination with anti-inflammatory steroid and/or ubiquinones is also provided. These agents and the compn. and formulations provided are suitable for the

treatment of respiratory tract, pulmonary and malignant diseases assocd. with bronchoconstriction, respiratory tract inflammation and **allergies**, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as **allergies**, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis, RDA, COPD, and cancers, among others.

The present agents and compn. may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L3 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:756484 CAPLUS
DOCUMENT NUMBER: 133:329593
TITLE: Low adenosine anti-sense oligonucleotide, compositions, kit and method for **treatment** of airway disorders associated with bronchoconstriction, lung inflammation, **allergy**(ies) and surfactant depletion
INVENTOR(S): Nyce, Jonathan W.
PATENT ASSIGNEE(S): East Carolina University, USA
SOURCE: PCT Int. Appl., 1592 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062736	A2	20001026	WO 2000-US8020	20000324
WO 2000062736	A3	20011011		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
BR 2000006019	A	20010313	BR 2000-6019	20000324
EP 1168919	A2	20020109	EP 2000-919668	20000324
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-127958P P 19990406
WO 2000-US8020 W 20000324
OTHER SOURCE(S): MARPAT 133:329593

AB An in vivo method of selectively delivering a nucleic acid to a target gene or mRNA, comprises the topical administration, e.g. to the respiratory system, of a subject of a therapeutic amt. of an oligonucleotide (oligo) that is antisense to the initiation codon region, the coding region, the 5' or 3' intron-exon junctions or regions within 2 to 10 nucleotides of the junctions of the gene or antisense to a mRNA complementary to the gene in an amt. effective to reach the target polynucleotide and reducing or inhibiting expression. In addn. a method of treating an adenosine-mediated effect comprises topically administering

to a subject an antisense oligo in an amt. effective to treat the respiratory, pulmonary, or airway disease. In order to minimize triggering adenosine receptors by their metab., the administered oligos have a low content of or are essentially free of adenosine. A pharmaceutical compn. and formulations comprise the oligo antisense to an adenosine receptor, genes and mRNAs encoding them, genomic and mRNA flanking regions, intron and exon borders and all regulatory and functionally related segments of the genes and mRNAs encoding the polypeptides, their salts and mixts. Various formulations contain a requisite carrier, and optionally other additives and biol. active agents.

The low-adenosine or adenosine-free (des-A) agent for practicing the method of the invention may be prepd. by selecting a target gene(s), genomic flanking region(s), RNA(s) and/or polypeptide(s) assocd. with a disease(s) or condition(s) afflicting lung airways, obtaining the sequence of the mRNA(s) corresponding to the target gene(s) and/or genomic flanking region(s), and/or RNAs encoding the target polypeptide(s), selecting at least one segment of the mRNA which may be up to 60 % free of thymidine (T) and synthesizing one or more anti-sense oligonucleotide(s) to the mRNA segments which are free of adenosine (A) by substituting a universal base for A when present in the oligonucleotide. The agent may be prepd. by selection of target nucleic acid sequences with GC running stretches, which have low T content, and by optionally replacing A in the antisense oligonucleotides with a "Universal or alternative base". The agent, compn. and formulations are used for prophylactic, preventive and therapeutic **treatment** of ailments assocd. with impaired respiration, lung **allergy**(ies) and/or inflammation and depletion lung surfactant or surfactant hypoprodn., such as pulmonary vasoconstriction, inflammation, **allergies**, allergic rhinitis, asthma, impeded respiration, lung pain, cystic fibrosis, bronchoconstriction. The present **treatment** is suitable for administration in combination with other **treatments**, e.g. before, during and after other **treatments**, including radiation, chemotherapy, antibody therapy and surgery, among others. Alternatively, the present agent is effectively administered prophylactically or therapeutically by itself for conditions without known therapies or as a substitute for therapies exhibiting undesirable side effects. The **treatment** of this invention may be administered directly into the respiratory system of a subject so that the agent has direct access to the lungs, or by other effective routes of administration, e.g. topically, transdermally, by implantation, etc., in an amt. effective to reduce or inhibit the symptoms of the ailment.

L3 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER:	2000:628006 CAPLUS
DOCUMENT NUMBER:	133:217723
TITLE:	Method for validating/invalidating target(s) and pathways
INVENTOR(S):	Nyce, Jonathan W.
PATENT ASSIGNEE(S):	Epigenesis Pharmaceuticals, Inc., USA
SOURCE:	PCT Int. Appl., 53 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000051621	A1	20000908	WO 2000-US5643	20000302
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
BR 2000009247	A	20011120	BR 2000-9247	20000302
EP 1165093	A1	20020102	EP 2000-913730	20000302
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002537792	T2	20021112	JP 2000-602288	20000302

PRIORITY APPLN. INFO.: US 1999-122950P P 19990305
WO 2000-US5643 W 20000302

OTHER SOURCE(S): MARPAT 133:217723

AB A method of detg. the existence of a correlation between a function of a disease or condition and a gene or mRNA encoding a target polypeptide suspected of being assocd. with a disease or condition, comprises obtaining oligonucleotides (oligos) consisting of up to about 15 % adenosine (A), preferably having no adenosine content, and which is anti-sense to a target selected from the group consisting of target genes and their corresponding mRNAs, genomic and mRNA flanking regions selected from the group consisting of 3' and 5' intron-exon borders and the juxta-section between coding and non-coding regions, and all mRNA segments encoding polypeptides assocd. with a pre-selected disease or condition; selecting amongst the oligos one that significantly inhibits or ablates expression of the polypeptide encoded by the mRNA upon in vitro hybridization to the target mRNA; administering to a subject an amt. of the selected oligo effective for in vivo hybridization to the target mRNA; and assessing a subject's function that is assocd. with the disease or condition before and after administration of the oligo; wherein a change in the function's value greater than about 70% indicates a pos. correlation, between about 40 and about 70% a possible correlation, and below about 30% a lack of correlation. The present method preferably administers the oligos in situ where the target is located, e.g. into the subject's respiration when validating targets assocd. with malignant and other pulmonary and respiratory functions, so that the agent has direct access to the lungs. Alternatively, such desAdenosine oligos may be delivered directly to the CNS or other organs, tissues and organ systems, by known delivery formulations. This invention provides a rapid, reliable method for drug target validation/invalidation in various biol. systems that utilize proprietary low or desAdenosine antisense oligonucleotides. Using desAdenosine antisense oligonucleotides, the present method may validate/invalidate potential gene targets with a level of speed and accuracy that has heretofore been impossible using traditional techniques.

The use of antisense oligonucleotides to target adenosine receptors is described. Adenosine A1 receptor antisense oligonucleotides had bronchodilator activity in rabbits and adenosine A3 receptor antisense oligonucleotides had anti-inflammatory activity in asthmatic rabbits.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:223049 CAPLUS
DOCUMENT NUMBER: 130:251233
TITLE: Macrophage-derived chemokine (MDC), MDC analogs, MDC inhibitor substances,and their therapeutic applications
INVENTOR(S): Gray, Patrick W.; Chantry, David H.; Deeley, Michael C.; Raport, Carol J.; Godiska, Ronald
PATENT ASSIGNEE(S): Icos Corporation, USA
SOURCE: PCT Int. Appl., 159 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9915666	A2	19990401	WO 1998-US20270	19980928
WO 9915666	A3	19990916		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, US, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CN 1163635	A	19971029	CN 1996-190875	19960607
US 5932703	A	19990803	US 1996-660542	19960607
CA 2302806	AA	19990401	CA 1998-2302806	19980928
AU 9897778	A1	19990412	AU 1998-97778	19980928
EP 1017818	A2	20000712	EP 1998-951961	19980928
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, SE, IE				

PRIORITY APPLN. INFO.:
US 1995-479620 A2 19950607
US 1995-558658 A2 19951116
US 1996-660542 A2 19960607
US 1997-939107 A2 19970926
US 1998-67447 A2 19980428
WO 1998-US20270 W 19980928

AB The present invention provides purified and isolated polynucleotide sequences encoding a novel macrophage-derived C-C chemokine designated "Macrophage Derived Chemokine" (MDC), and polypeptide fragments and analogs thereof. MDC cDNA sequences and their deduced amino acid sequences are provided from human, mouse, rat, and macaque. Also provided are materials and methods for the recombinant or synthetic prodn. of the chemokine, fragments, and analogs; and purified and isolated chemokine protein, and polypeptide fragments and analogs thereof. Also provided are antibodies reactive with the chemokine and methods of making and using all of the foregoing. Also provided are assays for identifying modulators of MDC chemokine activity. MDC possesses antiproliferative activity against HIV-1 virus, stimulates fibroblast proliferation, inhibits tumor growth, induces chemotaxis of TH2 helper T cells, and modulates platelet

PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 117 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001051631	A2	20010719	WO 2001-EP362	20010112
WO 2001051631	A3	20020418		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
DE 10001169	A1	20010726	DE 2000-10001169	20000113
EP 1250430	A2	20021023	EP 2001-903644	20010112
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.:
DE 2000-10001169 A 20000113
DE 2000-10010188 A 20000302
WO 2001-EP362 W 20010112

AB The invention relates to regulatory sequences which impart a specific expression in dendritic cells. The regulatory sequences are isolated from the human fascin gene and also comprise, for example, promoter sequences. The invention also relates to recombinant nucleic acid mols. and vectors, which contain the regulatory sequences, and to preferred embodiments of the recombinant nucleic acid mols. and vectors, which code the antigens or immunoregulatory proteins. The invention addnl. relates to host cells, which contain the recombinant nucleic acid mols. or vectors, and to methods for the prodn. thereof. Addnl. embodiments relate to in-vitro methods for stimulating T cells and for producing T cell-stimulating dendritic cells, and to their formulation as medicaments. Addnl. medicaments are described which essentially relate to DNA vaccines and to gene-therapeutic medicaments, for example, for the immunization against and for the treatment of infectious diseases, tumors, **allergies**, Creutzfeldt-Jakob plaques or Alzheimer plaques. Addnl. inventive medicaments can be used for the targeted modulation of immune responses that is imparted by dendritic cells, for example, for treating autoimmune diseases or transplant rejection. Finally, the invention relates to different uses of the regulatory sequences. Cloning of the gene and anal. of the 5'-flanking region using a luciferase reporter gene are described.

L2 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:402803 CAPLUS
DOCUMENT NUMBER: 136:84180
TITLE: Chemokines, chemokine receptors and **allergy**
AUTHOR(S): Kaplan, Allen P.
CORPORATE SOURCE: Division of Pulmonary Diseases and Central Case Medicine and Allergy and, Medical University of South Carolina, Charleston, SC, USA

NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2304312	AA	19990325	CA 1998-2304312	19980917
AU 9893951	A1	19990405	AU 1998-93951	19980917
AU 752531	B2	20020919		
EP 1019065	A1	20000719	EP 1998-947089	19980917

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI

BR 9812650	A	20000822	BR 1998-12650	19980917

PRIORITY APPLN. INFO.:

US 1997-59160P	P	19970917
US 1998-93972	A	19980609
WO 1998-US19419	W	19980917

AB Antisense oligonucleotides carrying sequences that will allow them to bind to more than one mRNA in a target cell are described. Such oligonucleotides can be used as a single treatment for diseases having more than one contributing pathway. In particular, oligonucleotides effective against genes involved in the etiol. of respiratory disease are targeted. Preferably, the oligonucleotides are low in adenosine (.ltoreq.15%) and may have adenosines substituted with analogs. These oligonucleotides are targeted to high (G+C) sequences within mRNAs. Thus, phosphorothioate antisense oligonucleotide (HAdA1AS, 5'-gatggagggcggcatggcggg-3') designed for the adenosine A1 receptor is provided. HAdA1AS significantly and specifically reduces the in vivo response to adenosine challenge in a dose-dependent manner, is effective in protection against aeroallergen-induced bronchoconstriction (house dust mite), has an unexpected long-term duration of effect (8.3 days for both PC50 adenosine and resistance), and is free of side effects that might be toxic to the recipient. Such oligonucleotides may be used for treating a disease or condition assocd. with lung airway bronchoconstriction, inflammation, or **allergies**.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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aggregation, and is shown to be a high-affinity ligand for **CCR4**.

L3 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:219995 CAPLUS
DOCUMENT NUMBER: 130:306599
TITLE: Antisense oligonucleotides capable of binding to multiple targets and their use in the **treatment** of respiratory disease
INVENTOR(S): Nyce, Jonathan W.
PATENT ASSIGNEE(S): East Carolina University, USA
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9913886	A1	19990325	WO 1998-US19419	19980917
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2304312	AA	19990325	CA 1998-2304312	19980917
AU 9893951	A1	19990405	AU 1998-93951	19980917
AU 752531	B2	20020919		
EP 1019065	A1	20000719	EP 1998-947089	19980917
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
BR 9812650	A	20000822	BR 1998-12650	19980917

PRIORITY APPLN. INFO.:
US 1997-59160P P 19970917
US 1998-93972 A 19980609
WO 1998-US19419 W 19980917

AB Antisense oligonucleotides carrying sequences that will allow them to bind to more than one mRNA in a target cell are described. Such oligonucleotides can be used as a single **treatment** for diseases having more than one contributing pathway. In particular, oligonucleotides effective against genes involved in the etiol. of respiratory disease are targeted. Preferably, the oligonucleotides are low in adenosine (.ltoreq.15%) and may have adenosines substituted with analogs. These oligonucleotides are targeted to high (G+C) sequences within mRNAs. Thus, phosphorothioate antisense oligonucleotide (HAdA1AS, 5'-gatggagggcggcatggcggg-3') designed for the adenosine A1 receptor is provided. HAdA1AS significantly and specifically reduces the in vivo response to adenosine challenge in a dose-dependent manner, is effective in protection against aeroallergen-induced bronchoconstriction (house dust mite), has an unexpected long-term duration of effect (8.3 days for both PC50 adenosine and resistance), and is free of side effects that might be toxic to the recipient. Such oligonucleotides may be used for treating a disease or condition assocd. with lung airway, such as bronchoconstriction, inflammation, or **allergies**.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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YOU HAVE REQUESTED DATA FROM 12 ANSWERS - CONTINUE? Y/(N):Y
THE ESTIMATED COST FOR THIS REQUEST IS 27.47 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L2 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:549831 CAPLUS
DOCUMENT NUMBER: 137:139275
TITLE: Nickel-specific CD4+ and CD8+ T cells display distinct migratory responses to chemokines produced during allergic contact dermatitis
AUTHOR(S): Sebastiani, Silvia; Albanesi, Cristina; Nasorri, Francesca; Girolomoni, Giampiero; Cavani, Andrea
CORPORATE SOURCE: Laboratory of Immunology, Istituto Dermopatico dell'Immacolata, IRCCS, Rome, 00167, Italy
SOURCE: Journal of Investigative Dermatology (2002), 118(6), 1052-1058
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Development of allergic contact dermatitis to haptens depends upon a balance between CD8+ T lymphocytes with pathogenic activity and CD4+ T cells, which comprise both effector and regulatory cells. Thus, differential recruitment of CD8+ and CD4+ lymphocytes to sites of hapten challenge may have considerable impact on disease expression. Here the migration of cutaneous lymphocyte-assocd. antigen+, nickel-specific CD8+ and CD4+ T cell lines were compared with a panel of chemokines produced in the skin during allergic contact dermatitis. CCL17/TARC and CCL22/**MDC** induced a 3-fold higher migration of CD4+ compared with CD8+ lymphocytes. In contrast, CXCL10/IP-10 was 2-fold more potent in attracting CD8+ cells. These findings were consistent with the higher expression of CCR4 and CXCR3 on CD4+ and CD8+ T cell lines, resp. Moreover, CCR4 expression was high on nickel-specific T helper 2, intermediate on T helper 1 and T cytotoxic 2, and almost undetectable on T cytotoxic 1 clones. On the contrary, CXCR3 was expressed by T cytotoxic 1 and 2 and T helper 1, but not T helper 2 clones. Reverse transcription-polymerase chain reaction anal. of the skin before and after hapten challenge revealed the constitutive presence of TARC, and the early appearance of CCL2/MCP-1, followed by IP-10, CCL4/MIP-1.beta., and **MDC** mRNA. Supernatants from activated keratinocytes induced a strong migration of CD8+ lymphocytes, which was blocked by neutralization of IP-10. Conversely, supernatants from immature and mature dendritic cells attracted mostly CD4+ lymphocytes in a TARC- and **MDC**-dependent manner. Our data indicate that distinct chemokines and cell types control the accumulation of CD8+ and CD4+ T cells within inflamed skin.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:479577 CAPLUS
DOCUMENT NUMBER: 137:92625
TITLE: Human epithelial cells trigger dendritic cell-mediated allergic inflammation by producing TSLP
AUTHOR(S): Soumelis, Vassili; Reche, Pedro A.; Kanzler, Holger; Yuan, Wei; Edward, Gina; Homey, Bernhart; Gilliet, Michel; Ho, Steve; Antonenko, Svetlana; Lauerma, Annti; Smith, Kathleen; Gorman, Daniel; Zurawski, Sandra; Abrams, Jon; Menon, Satish; McClanahan, Terri; de Waal-Malefyt, Rene; Bazan, Fernando; Kastelein, Robert A.; Liu, Yong-Jun
CORPORATE SOURCE: DNAX, Palo Alto, CA, 94304, USA
SOURCE: Nature Immunology (2002), 3(7), 673-680
CODEN: NIAMCZ; ISSN: 1529-2908
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Whether epithelial cells play a role in triggering the immune cascade leading to T helper 2 (TH2)-type allergic inflammation is not known. We show here that human thymic stromal lymphopoietin (TSLP) potently activated CD11c+ dendritic cells (DCs) and induced prodn. of the TH2-attracting chemokines TARC (thymus and activation-regulated chemokine; also known as CCL17) and **MDC** (macrophage-derived chemokine; CCL22). TSLP-activated DCs primed naive TH cells to produce the proallergic cytokines interleukin 4 (IL-4), IL-5, IL-13 and tumor necrosis factor-.alpha., while down-regulating IL-10 and interferon-.gamma.. TSLP was highly expressed by epithelial cells, esp. keratinocytes from patients with atopic dermatitis. TSLP expression was assocd. with Langerhans cell migration and activation in situ. These findings shed new light on the function of human TSLP and the role played by epithelial cells and DCs in initiating allergic inflammation.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:429201 CAPLUS
DOCUMENT NUMBER: 137:4997
TITLE: Method for diagnosing allergic diseases using DNA and protein microarray technology
INVENTOR(S): Schmidt-Weber, Carsten; Blaser, Kurt; Wohlfahrt, Jan
PATENT ASSIGNEE(S): Genescan Europe Ag, Germany
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002044732	A2	20020606	WO 2001-EP13937	20011129

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1221618 A1 20020710 EP 2000-126117 20001129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
AU 2002021906 A5 20020611 AU 2002-21906 20011129
PRIORITY APPLN. INFO.: EP 2000-126117 A 20001129
WO 2001-EP13937 W 20011129

AB MRNA of activated lymphocytes such as CD4+ T cells allows differential diagnosis of allergic diseases. The CD4+ T cells are isolated and stimulated under defined conditions in vitro. Subsequently, mRNA is subjected to multigene anal. such as DNA arrays. Expression profiling images, such as gene expression profiles, can be created, which allow on the basis of the activated T cell mRNA the prediction of certain phenotypes such as asthma or atopic dermatitis.

L2 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:354656 CAPLUS
DOCUMENT NUMBER: 137:31703
TITLE: Cytokines and chemoattractants in allergic inflammation
AUTHOR(S): Romagnani, S.
CORPORATE SOURCE: Department of Internal Medicine, and Respiratory Diseases, Allergy, Section of Clinical Immunology, University of Florence, Florence, 50134, Italy
SOURCE: Molecular Immunology (2002), 38(12-13), 881-885
CODEN: MOIMD5; ISSN: 0161-5890
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. It is now generally accepted that type 2 T helper (Th2) cytokines and some chemoattractants play an essential role in the pathogenesis of the allergic inflammation. The effects of Th2 cytokines, such as interleukin (IL)-4, IL-5, IL-9, and IL-13, account for virtually all the pathophysiol. manifestations of **allergy** and asthma. Moreover, both Th2 cells and the effector cells usually present in the areas of allergic inflammation (basophils, mast cells, and eosinophils) express chemoattractant receptors, such as CCR3, CCR4, CCR8, and CRTH2. Therefore, interactions of eotaxin(s), eotaxin/CCL11, RANTES/CCL5, and MCP-1/CCL2, MCP-2/CCL8, MCP-3/CCL7, MCP-4/CCL13 with CCR3 are responsible for the recruitment of basophils, eosinophils and mast cells, whereas interactions of CCR4 with **MDC**/CCL22 or TARC/CCL17, CCR8 with I-309/CCL1, and CRTH2 with PGD2 play a crit. role in the allergen-induced recruitment of Th2 cells in the target tissues of allergic inflammation. The demonstration that Th2-polarized responses against allergens represent the triggering event for the development of allergic diseases, together with the recognition that some chemoattractants are responsible for the recruitment of both Th2 cells and other effector cells of allergic inflammation, can provide the conceptual basis for the development of new therapeutic strategies in allergic conditions.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L2 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:693651 CAPLUS
DOCUMENT NUMBER: 135:240908
TITLE: Assay for agents that induce chemokinesis
INVENTOR(S): Carson, Dennis A.; Leoni, Lorenzo M.; Cottam, Howard B.
PATENT ASSIGNEE(S): Regents of the University of California, USA
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001069240	A1	20010920	WO 2001-US8581	20010316

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002010125	A1	20020124	US 2001-810010	20010316

PRIORITY APPLN. INFO.: US 2000-189976P P 20000316
AB The present invention provides methods for identifying compds. that can induce cellular chemokinesis. According to the present invention, chemokinesis interferes with immune and inflammatory responses by increasing cell movements and altering cell migration patterns. Surprisingly, compds. isolated according to the present invention can interfere with the spread of malignant cells through the body, reduce inflammatory responses and can cause leukocytes to be retained in lymph nodes, the spleen and other organs of the reticulo-endothelial system. Several methods are contemplated by the present invention for identifying compds. which can induce chemokinesis. In one embodiment the method involves contacting a population of target cells with a test compd. and observing whether the target cells produce a chemotactic mol.; wherein the target cell has a cognate receptor for the chemotactic mol. In another embodiment, the method involves contacting a population of target cells with a test compd. and observing whether the targets cells homotypically aggregate. In yet another embodiment, the method involves contacting a population of target cells with a test compd. and observing whether actin filaments in the target cells form stress fibers.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:526192 CAPLUS
DOCUMENT NUMBER: 135:117975
TITLE: Regulatory sequence for dendritic cell-specific expression from human fascin genes and their use
INVENTOR(S): Reske-Kunz, Angelika; Ross, Xiaolan; Ross, Ralf; Bros, Matthias

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, US, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CN 1163635	A	19971029	CN 1996-190875	19960607
US 5932703	A	19990803	US 1996-660542	19960607
CA 2302806	AA	19990401	CA 1998-2302806	19980928
AU 9897778	A1	19990412	AU 1998-97778	19980928
EP 1017818	A2	20000712	EP 1998-951961	19980928

R: AT, BE, CH, DE, ES, FR, GB, IT, LI, SE, IE

PRIORITY APPLN. INFO.:	US 1995-479620	A2	19950607
	US 1995-558658	A2	19951116
	US 1996-660542	A2	19960607
	US 1997-939107	A2	19970926
	US 1998-67447	A2	19980428
	WO 1998-US20270	W	19980928

AB The present invention provides purified and isolated polynucleotide sequences encoding a novel macrophage-derived C-C chemokine designated "Macrophage Derived Chemokine" (MDC), and polypeptide fragments and analogs thereof. MDC cDNA sequences and their deduced amino acid sequences are provided from human, mouse, rat, and macaque. Also provided are materials and methods for the recombinant or synthetic prodn. of the chemokine, fragments, and analogs; and purified and isolated chemokine protein, and polypeptide fragments and analogs thereof. Also provided are antibodies reactive with the chemokine and methods of making and using all of the foregoing. Also provided are assays for identifying modulators of MDC chemokine activity. MDC possesses antiproliferative activity against HIV-1 virus, stimulates fibroblast proliferation, inhibits tumor growth, induces chemotaxis of TH2 helper T cells, and modulates platelet aggregation, and is shown to be a high-affinity ligand for **CCR4**.

L1 ANSWER 20 OF 20 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER:	1999:219995 CAPLUS
DOCUMENT NUMBER:	130:306599
TITLE:	Antisense oligonucleotides capable of binding to multiple targets and their use in the treatment of respiratory disease
INVENTOR(S):	Nyce, Jonathan W.
PATENT ASSIGNEE(S):	East Carolina University, USA
SOURCE:	PCT Int. Appl., 120 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9913886	A1	19990325	WO 1998-US19419	19980917

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,

DOCUMENT NUMBER: 132:48648
TITLE: Chemokines fundamental to the inflammation associated with allergic disorders
AUTHOR(S): Hirai, Koichi
CORPORATE SOURCE: Dep. of Bioregul. and Funct., Univ. of Tokyo Grad. Sch. of Med., Japan
SOURCE: Saishin Igaku (1999), 54(12), 2884-2887
CODEN: SAIGAK; ISSN: 0370-8241
PUBLISHER: Saishin Igakusha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review, with 19 refs., on expression of chemokine receptors on eosinophils, increased expression of eotaxin in allergic inflammation sites of human, cytokines regulating eotaxin expression, and CCR3 as a single receptor for eotaxin. Attempts of therapy for allergic inflammation by inhibiting TARC (thymus- and activation-regulated chemokine) and **MDC** (macrophage-derived chemokine) on Th2 cells are also discussed.

L2 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:223049 CAPLUS
DOCUMENT NUMBER: 130:251233
TITLE: Macrophage-derived chemokine (**MDC**), **MDC** analogs, **MDC** inhibitor substances,and their therapeutic applications
INVENTOR(S): Gray, Patrick W.; Chantry, David H.; Deeley, Michael C.; Raport, Carol J.; Godiska, Ronald
PATENT ASSIGNEE(S): Icos Corporation, USA
SOURCE: PCT Int. Appl., 159 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9915666	A2	19990401	WO 1998-US20270	19980928
WO 9915666	A3	19990916		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, US, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CN 1163635	A	19971029	CN 1996-190875	19960607
US 5932703	A	19990803	US 1996-660542	19960607
CA 2302806	AA	19990401	CA 1998-2302806	19980928
AU 9897778	A1	19990412	AU 1998-97778	19980928
EP 1017818	A2	20000712	EP 1998-951961	19980928
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, SE, IE				

PRIORITY APPLN. INFO.:

US 1995-479620	A2	19950607
US 1995-558658	A2	19951116
US 1996-660542	A2	19960607
US 1997-939107	A2	19970926
US 1998-67447	A2	19980428
WO 1998-US20270	W	19980928

AB The present invention provides purified and isolated polynucleotide sequences encoding a novel macrophage-derived C-C chemokine designated "Macrophage Derived Chemokine" (**MDC**), and polypeptide fragments and analogs thereof. **MDC** cDNA sequences and their deduced amino acid sequences are provided from human, mouse, rat, and macaque. Also provided are materials and methods for the recombinant or synthetic prodn.
of the chemokine, fragments, and analogs; and purified and isolated chemokine protein, and polypeptide fragments and analogs thereof. Also provided are antibodies reactive with the chemokine and methods of making and using all of the foregoing. Also provided are assays for identifying modulators of **MDC** chemokine activity. **MDC** possesses antiproliferative activity against HIV-1 virus, stimulates fibroblast proliferation, inhibits tumor growth, induces chemotaxis of TH2 helper T cells, and modulates platelet aggregation, and is shown to be a high-affinity ligand for CCR4.

=> DIS L1 1- IBIB ABS
YOU HAVE REQUESTED DATA FROM 20 ANSWERS - CONTINUE? Y/(N):Y
THE ESTIMATED COST FOR THIS REQUEST IS 45.78 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1 ANSWER 1 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:832576 CAPLUS
TITLE: Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 764 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085309	A2	20021031	WO 2002-US13143	20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-286036P P 20010424
AB This patent relates to a compn. comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting

human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addn., they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the treatment of bronchoconstriction and/or inflammation. These agents and the compn. and formulations provided are suitable for the treatment of respiratory tract, pulmonary and malignant diseases assocd. with bronchoconstriction, respiratory tract inflammation and **allergies**, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as **allergies**, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis, RDA, COPD, and cancers, among others. The present agents and compn. may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L1 ANSWER 2 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:832575 CAPLUS
TITLE: Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 872 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085308	A2	20021031	WO 2002-US13135	20020423
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002085308	A2	20021031	WO 2002-XA13135	20020423
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,				

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002085308 A2 20021031 WO 2002-XB13135 20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002085308 A2 20021031 WO 2002-XC13135 20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-286137P P 20010424
WO 2002-US13135 A 20020423

AB This patent relates to a compn. comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addn., they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the treatment of bronchoconstriction and/or inflammation. Treatment with antisense oligonucleotides in combination with anti-inflammatory steroid and/or ubiquinones is also provided. These agents and the compn. and formulations provided are suitable for the treatment of respiratory tract, pulmonary and malignant diseases assocd. with bronchoconstriction, respiratory tract inflammation and **allergies**, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as **allergies**, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis,

RDA, COPD, and cancers, among others. The present agents and compn. may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L1 ANSWER 3 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:571773 CAPLUS
DOCUMENT NUMBER: 137:153743
TITLE: Airway hyperresponsiveness, but not airway remodeling, is attenuated during chronic pulmonary allergic responses to Aspergillus in **CCR4**-/- mice
AUTHOR(S): Schuh, Jane M.; Power, Christine A.; Proudfoot, Amanda E.; Kunkel, Steven L.; Lukacs, Nicholas W.; Hogaboam, Cory M.
CORPORATE SOURCE: Department of Pathology, University of Michigan Medical School, Ann Arbor, MI, USA
SOURCE: FASEB Journal (2002), 16(10), 1313-1315, 10.1096/fj.02-0193fje
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The role of CC chemokine receptor 4 (**CCR4**) during the development and maintenance of Th2- type allergic airway disease is controversial. In this study, we examd. the role of **CCR4** in the chronic allergic airway response to live Aspergillus fumigatus spores, or conidia, in A. fumigatus-sensitized mice. After the conidia challenge, mice lacking **CCR4** (**CCR4**-/- mice) exhibited significantly increased nos. of airway neutrophils and macrophages, and conidia were more rapidly eliminated from these mice compared with control **CCR4** wild-type (**CCR4**+/+) mice. Significant airway hyperresponsiveness to i.v. methacholine was obsd. at day 3 in **CCR4**-/- mice, whereas at days 7 and 30, airway hyperresponsiveness was attenuated in these mice compared with control mice. A major redn. in peribronchial and airway eosinophilia was obsd. in **CCR4**-/- mice at all times after conidia challenge in contrast to **CCR4**+/+ mice. Further, whole lung levels of interleukin (IL) 4 and IL-5 were significantly increased in **CCR4**-/- mice at day 3, whereas these Th2 cytokines and IL-13 were significantly decreased at day 30 in **CCR4**-/- mice compared with their wild-type counterparts. Peribronchial fibrosis and goblet cell hyperplasia were similar in both groups of mice throughout the course of this model. In summary, **CCR4** modulates both innate and acquired immune responses assocd. with chronic fungal asthma.
REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 4 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:549831 CAPLUS
DOCUMENT NUMBER: 137:139275

TITLE: Nickel-specific CD4+ and CD8+ T cells display distinct migratory responses to chemokines produced during allergic contact dermatitis
AUTHOR(S): Sebastiani, Silvia; Albanesi, Cristina; Nasorri, Francesca; Girolomoni, Giampiero; Cavani, Andrea
CORPORATE SOURCE: Laboratory of Immunology, Istituto Dermopatico dell'Immacolata, IRCCS, Rome, 00167, Italy
SOURCE: Journal of Investigative Dermatology (2002), 118(6), 1052-1058
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Development of allergic contact dermatitis to haptens depends upon a balance between CD8+ T lymphocytes with pathogenic activity and CD4+ T cells, which comprise both effector and regulatory cells. Thus, differential recruitment of CD8+ and CD4+ lymphocytes to sites of hapten challenge may have considerable impact on disease expression. Here the migration of cutaneous lymphocyte-assocd. antigen+, nickel-specific CD8+ and CD4+ T cell lines were compared with a panel of chemokines produced in the skin during allergic contact dermatitis. CCL17/TARC and CCL22/MDC induced a 3-fold higher migration of CD4+ compared with CD8+ lymphocytes. In contrast, CXCL10/IP-10 was 2-fold more potent in attracting CD8+ cells. These findings were consistent with the higher expression of **CCR4** and CXCR3 on CD4+ and CD8+ T cell lines, resp. Moreover, **CCR4** expression was high on nickel-specific T helper 2, intermediate on T helper 1 and T cytotoxic 2, and almost undetectable on T cytotoxic 1 clones. On the contrary, CXCR3 was expressed by T cytotoxic 1 and 2 and T helper 1, but not T helper 2 clones. Reverse transcription-polymerase chain reaction anal. of the skin before and after hapten challenge revealed the constitutive presence of TARC, and the early appearance of CCL2/MCP-1, followed by IP-10, CCL4/MIP-1.beta., and MDC mRNA. Supernatants from activated keratinocytes induced a strong migration of CD8+ lymphocytes, which was blocked by neutralization of IP-10. Conversely, supernatants from immature and mature dendritic cells attracted mostly CD4+ lymphocytes in a TARC- and MDC-dependent manner. Our data indicate that distinct chemokines and cell types control the accumulation of CD8+ and CD4+ T cells within inflamed skin.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 5 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:354656 CAPLUS
DOCUMENT NUMBER: 137:31703
TITLE: Cytokines and chemoattractants in allergic inflammation
AUTHOR(S): Romagnani, S.
CORPORATE SOURCE: Department of Internal Medicine, and Respiratory Diseases, Allergy, Section of Clinical Immunology, University of Florence, Florence, 50134, Italy
SOURCE: Molecular Immunology (2002), 38(12-13), 881-885
CODEN: MOIMD5; ISSN: 0161-5890
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review

LANGUAGE: English
AB A review. It is now generally accepted that type 2 T helper (Th2) cytokines and some chemoattractants play an essential role in the pathogenesis of the allergic inflammation. The effects of Th2 cytokines, such as interleukin (IL)-4, IL-5, IL-9, and IL-13, account for virtually all the pathophysiol. manifestations of **allergy** and asthma. Moreover, both Th2 cells and the effector cells usually present in the areas of allergic inflammation (basophils, mast cells, and eosinophils) express chemoattractant receptors, such as CCR3, **CCR4**, CCR8, and CRTH2. Therefore, interactions of eotaxin(s), eotaxin/CCL11, RANTES/CCL5, and MCP-1/CCL2, MCP-2/CCL8, MCP-3/CCL7, MCP-4/CCL13 with CCR3 are responsible for the recruitment of basophils, eosinophils and mast cells, whereas interactions of **CCR4** with MDC/CCL22 or TARC/CCL17, CCR8 with I-309/CCL1, and CRTH2 with PGD2 play a crit. role in the allergen-induced recruitment of Th2 cells in the target tissues of allergic inflammation. The demonstration that Th2-polarized responses against allergens represent the triggering event for the development of allergic diseases, together with the recognition that some chemoattractants are responsible for the recruitment of both Th2 cells and other effector cells of allergic inflammation, can provide the conceptual basis for the development of new therapeutic strategies in allergic conditions.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 6 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:304568 CAPLUS
DOCUMENT NUMBER: 137:167744
TITLE: The role of TARC in the pathogenesis of allergic asthma
AUTHOR(S): Berin, M. Cecilia
CORPORATE SOURCE: Division of Pediatric Allergy and Immunology, Mount Sinai School of Medicine, New York, NY, USA
SOURCE: Drug News & Perspectives (2002), 15(1), 10-16
CODEN: DNPEED; ISSN: 0214-0934
PUBLISHER: Prous Science
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. TARC (thymus and activation-regulated chemokine), as a selective chemoattractant of Th2 cells, is a reasonable candidate as a key regulator of Th2-mediated inflammation in allergic asthma. Studies have detd. that TARC is up-regulated in the airways of human subjects with asthma and that **CCR4**- and CCR8-bearing T cells are also present in the airways of asthmatic subjects after allergen challenge. Mouse models of allergic airway inflammation have shown that neutralization of TARC can not only inhibit T-cell and eosinophil infiltration into the lung but can also inhibit bronchial hyperresponsiveness. The exact mechanism by which TARC can participate in allergic inflammation and what triggers the expression of TARC following allergen exposure is still unknown. Studies suggest that it could be involved not only in allergic asthma, but in the pathogenesis of allergic Th2-mediated diseases in general.
REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L1 ANSWER 7 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:780964 CAPLUS
DOCUMENT NUMBER: 135:330487
TITLE: Uses of interleukin 174 agonists and antagonists
INVENTOR(S): Hurst, Stephen D.; Zurawski, Sandra M.; Rennick, Donna
M.
PATENT ASSIGNEE(S): Schering Corporation, USA
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001079288	A2	20011025	WO 2001-US12493	20010417
WO 2001079288	A3	20020510		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-198488P P 20000418
AB Agonists or antagonists of the cytokine designated IL-174, and various methods of their use are provided. In particular, the methods make use of facts that many activities of the IL-174 cytokine are described. The agonists and antagonists may be used in therapy of many different types of diseases and infections.

L1 ANSWER 8 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:709559 CAPLUS
DOCUMENT NUMBER: 136:323434
TITLE: Chemoattractant Receptors Expressed on Type 2 T Cells and Their Role in Disease
AUTHOR(S): Cosmi, Lorenzo; Annunziato, Francesco; Maggi, Enrico; Romagnani, Sergio; Manetti, Roberto
CORPORATE SOURCE: Department of Internal Medicine, Section of Clinical Immunology, Allergy and Respiratory Diseases, University of Florence, Italy
SOURCE: International Archives of Allergy and Immunology (2001), 125(4), 273-279
CODEN: IAAIEG; ISSN: 1018-2438
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The existence of two functionally distinguished populations among T cells has been established in both mice and humans. Type 1 T helper (Th1) cells are involved in the defense against intracellular bacteria and many viruses, while type 2 Th cells (Th2) are the major

actors in the response against parasites and play a central role in allergic inflammation. More recently, several data have suggested that some chemokine receptors are tightly regulated on T cells, and in accordance with this selective expression, Th1 and Th2 cells can be differentially recruited by specific chemokines to the inflammatory sites.

Among Th2-assocd. chemokine receptors, CCR3, **CCR4** and CCR8 have been described to play a central role in allergic inflammation. However, CCR3 is mainly expressed on basophils, eosinophils and mast cells, but it is poorly expressed by Th2 cells, and **CCR4** is also expressed by Th subsets different from Th2 cells. So far, the chemoattractant receptors which among T cells appear to be selectively expressed by Th2 cells or their subsets are CCR8 and CRTH2. The ligand for CRTH2 is not a chemokine, but is prostaglandin (PG)D2, which is able to attract basophils, eosinophils, Th2 cells and type 2 cytotoxic (Tc2) CD8+ T lymphocytes. The selective expression of CRTH2 on Th2 and Tc2 cells may be useful to develop new therapeutic strategies against allergic diseases and against other immune disorders. Addnl. studies, however, are required to understand its effective importance in the induction and maintenance of Th2- or Tc2-mediated response and inflammation.

REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 9 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:693651 CAPLUS
DOCUMENT NUMBER: 135:240908
TITLE: Assay for agents that induce chemokinesis
INVENTOR(S): Carson, Dennis A.; Leoni, Lorenzo M.; Cottam, Howard B.
PATENT ASSIGNEE(S): Regents of the University of California, USA
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001069240	A1	20010920	WO 2001-US8581	20010316
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002010125	A1	20020124	US 2001-810010	20010316

PRIORITY APPLN. INFO.: US 2000-189976P P 20000316

AB The present invention provides methods for identifying compds. that can induce cellular chemokinesis. According to the present invention, chemokinesis interferes with immune and inflammatory responses by increasing cell movements and altering cell migration patterns. Surprisingly, compds. isolated according to the present invention can

interfere with the spread of malignant cells through the body, reduce inflammatory responses and can cause leukocytes to be retained in lymph nodes, the spleen and other organs of the reticulo-endothelial system. Several methods are contemplated by the present invention for identifying compds. which can induce chemokinesis. In one embodiment the method involves contacting a population of target cells with a test compd. and observing whether the target cells produce a chemotactic mol.; wherein the target cell has a cognate receptor for the chemotactic mol. In another embodiment, the method involves contacting a population of target cells with a test compd. and observing whether the targets cells homotypically aggregate. In yet another embodiment, the method involves contacting a population of target cells with a test compd. and observing whether actin filaments in the target cells form stress fibers.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 10 OF 20 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:402803 CAPLUS

DOCUMENT NUMBER: 136:84180

TITLE: Chemokines, chemokine receptors and **allergy**

AUTHOR(S): Kaplan, Allen P.

CORPORATE SOURCE: Division of Pulmonary Diseases and Central Case Medicine and Allergy and, Medical University of South Carolina, Charleston, SC, USA

SOURCE: International Archives of Allergy and Immunology (2001), 124(4), 423-431
CODEN: IAAIEG; ISSN: 1018-2438

PUBLISHER: S. Karger AG

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Chemokines are a group of cytokines that are responsible for the influx of blood cells, including T and B lymphocytes, monocytes, neutrophils, eosinophils and basophils, in allergic and other inflammatory conditions. They function as G protein-coupled chemotactic factors which also activate the cells with which they interact. Certain chemokines function within the afferent arm of the immune system, in which antigen is processed and antibody formation initiated, and others are active within the effector pathways of cellular immunity and late-phase allergic reactions. Th2 lymphocytes, which are crit. for **allergy**, employ the CC chemokine receptors **CCR4** and CCR8 with the ligands thymus- and activation-regulated chemokine (TARC), macrophage-derived chemokine (MDC) and I-309, resp. The chemokine receptor CCR3 and ligands monocyte chemoattractant protein (MCP)-3, MCP-4, regulated upon activation normal T cell expressed and secreted (RANTES) and eotaxins I and II are of particular relevance for the recruitment and activation of eosinophils. Th1 reactions depend upon interferon .gamma.-induced CXC chemokines interferon-inducible protein (IP)-10, interferon-inducible T cell-.alpha. chemoattractant (iTAC) and monokine induced by interferon-.gamma. (MiG), which bind to chemokine receptor CXCR3.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 11 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:340634 CAPLUS
DOCUMENT NUMBER: 135:70896
TITLE: Inhibition of **CCR4** ligands production by oriental medicines
AUTHOR(S): Hirai, Koichi; Nakajima, Toshiharu; Cyong, Jong Chol
CORPORATE SOURCE: Dep. Bioregul. Funct., Univ. Tokyo, Grad. Sch. Med., Tokyo, Japan
SOURCE: Kanpo to Men'eki, Arerugi (2000), 14, 121-129
CODEN: KMARED; ISSN: 0914-6407
PUBLISHER: Fama Intanashionaru
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Allergic inflammation is a Th2-dominant immune reaction. Thymus- and activation-regulated chemokine (TARC) is a ligand specific to the receptor **CCR4**, which is preferentially expressed Th2, there by being supposed to participate in the development of allergic inflammation. TARC derived from respiratory epithelial cells play a closely connected part in the pathogenesis of airway **allergy** through chemoattraction of Th2 cells. This study uses respiratory epithelial cells to investigate the effect of oriental medical prepns. on TARC prodn. Large quantities (ng/mL order) of TARC were produced in the supernatant of cultures of A549 human respiratory epithelial cells co-stimulated by TNF-.alpha. and IL-4. Thirteen oriental prepns., including Ogon (Huang-Qin) and Oren (Huang-Lian), were shown to inhibit TARC prodn., with Mao (Ma-Huang) demonstrating esp. strong inhibition action. It inhibited accumulation of TARC mRNA indicating that Mao inhibits TARC prodn. at the level of transcription. Ephedrine and pseudoephedrine, major components of Mao, also inhibited TARC prodn. at the protein and mRNA levels.

L1 ANSWER 12 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:300895 CAPLUS
DOCUMENT NUMBER: 134:279681
TITLE: Process for producing polypeptide
INVENTOR(S): Ogawa, Tatsuya; Konno, Yoshinobu; Akashi, Naohisa; Takasugi, Hiroshi; Sugimoto, Seiji; Yano, Keiichi
PATENT ASSIGNEE(S): Kyowa Hakko Kogyo Co., Ltd., Japan
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001029246	A1	20010426	WO 2000-JP7288	20001019

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,

CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 2000079504 A5 20010430 AU 2000-79504 20001019
EP 1229125 A1 20020807 EP 2000-969908 20001019
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL
PRIORITY APPLN. INFO.: JP 1999-296267 A 19991019
WO 2000-JP7288 W 20001019

AB A process for producing a desired polypeptide by using rat cells. More particularly speaking, a process for producing the polypeptide which comprises culturing rat cells such as YB2/3HL.P2.G11.16Ag.20 (hereinafter referred to as YB2/0), preferably rat cells obtained by transferring a recombinant DNA contg. a DNA encoding the desired polypeptide such as an immunol. functional mol., in a serum-free medium. Among the desired polypeptides obtained by this method, an antibody obtained by, for example, using transformants of YB2/0 has a high antibody-dependent cytotoxic activity and thus is useful as drugs.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L1 ANSWER 13 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:125162 CAPLUS
DOCUMENT NUMBER: 134:294296
TITLE: T cell phenotypes of the normal nasal mucosa: induction of Th2 cytokines and CCR3 expression by IL-4
AUTHOR(S): Till, Stephen J.; Jopling, Louise A.; Wachholz, Petra A.; Robson, Rachel L.; Qin, Shixin; Andrew, David P.; Wu, Lijun; Van Neerven, Joost; Williams, Timothy J.; Durham, Stephen R.; Sabroe, Ian
CORPORATE SOURCE: Upper Respiratory Medicine, National Heart and Lung Institute Division, Biomedical Sciences Division, Imperial College School of Medicine, London, UK
SOURCE: Journal of Immunology (2001), 166(4), 2303-2310
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mucosal environments such as that of the nose are points of first contact between the human organism and its environment. At these sites the immune system must be regulated to differentiate between and respond appropriately to pathogens and harmless contaminants. T cell-driven immune responses broadly fall into Th1- or Th2-type phenotypes, with increasing evidence that the recruitment of these T lymphocyte subsets is mediated by selective expression of specific chemokine receptors. We have investigated the immunol. of the normal nasal mucosa. We show that nasal T cell lines from normal individuals, expanded by culture in IL-2, show reduced expression of the Th2-type cytokines IL-4 and IL-5 compared with lines derived from the blood of the same subjects. These T cells also show reduced expression of the Th2-selective chemokine receptor, CCR3, but similar levels of **CCR4** compared with the blood-derived lines. This apparent suppression of Th2 cytokine and CCR3 expression by nasal T cells was reversed by addn. of IL-4 to the culture medium. These data are consistent with the presence of a nasal mucosal microenvironment that suppresses Th2 responses and may represent a protective measure against

atopic allergic disease in humans and a favoring of Th1 responses to infectious agents. In contrast, T cell expression of CCR1 was higher in the nose than in the blood regardless of the culture medium cytokine environment in keeping with a role for this receptor in tissue homing or lymphocyte activation.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 14 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:122695 CAPLUS
DOCUMENT NUMBER: 135:225348
TITLE: Chemokines and allergic diseases
AUTHOR(S): Adachi, Yuichi; Yamamoto, Junko; Miyawaki, Toshio
CORPORATE SOURCE: School of Medicine, Department of Pediatrics, Toyama Medical and Pharmaceutical University, Japan
SOURCE: Molecular Medicine (Tokyo) (2001), 38(2), 160-166
CODEN: MOLMEL; ISSN: 0918-6557
PUBLISHER: Nakayama Shoten
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review with 26 refs. on the role of eotaxin-CCR3 chemokines in the aggregation of inflammatory cells in **allergies**, chemokines in **allergies**, and the movement of T cell subsets (**CCR4**-pos. T cells, Th1 cells, Th2 cells) in **allergies**.

L1 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:100131 CAPLUS
DOCUMENT NUMBER: 134:251122
TITLE: Intervention of thymus and activation-regulated chemokine attenuates the development of allergic airway inflammation and hyperresponsiveness in mice
AUTHOR(S): Kawasaki, Shin; Takizawa, Hajime; Yoneyama, Hiroyuki; Nakayama, Takashi; Fujisawa, Ryuichi; Izumizaki, Masahiko; Imai, Toshio; Yoshie, Osamu; Homma, Ikuo; Yamamoto, Kazuhiko; Matsushima, Kouji
CORPORATE SOURCE: Department of Respiratory Medicine, Molecular Preventive Medicine, School of Medicine, University of Tokyo, Tokyo, Japan
SOURCE: Journal of Immunology (2001), 166(3), 2055-2062
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thymus- and activation-regulated chemokine (TARC; CCL17) is a lymphocyte-directed CC chemokine that specifically chemoattracts CC chemokine receptor 4-pos. (**CCR4**+) Th2 cells. To establish the pathophysiol. roles of TARC in vivo, we investigated here whether an mAb against TARC could inhibit the induction of asthmatic reaction in mice elicited by OVA. TARC was constitutively expressed in the lung and was up-regulated in allergic inflammation. The specific Ab against TARC attenuated OVA-induced airway eosinophilia and diminished the degree of airway hyperresponsiveness with a concomitant decrease in Th2 cytokine levels. Our results for the first time indicate that TARC is a pivotal chemokine for the development of Th2-dominated exptl. allergen-induced asthma with eosinophilia and AHR. This study also represents the first success in controlling Th2 cytokine prodn. in vivo by targeting a chemokine.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L1 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:903436 CAPLUS
DOCUMENT NUMBER: 135:120758
TITLE: Non-redundant functional groups of chemokines operate in a coordinate manner during the inflammatory response in the lung
AUTHOR(S): Gutierrez-Ramos, J. -C.; Lloyd, C.; Kapsenberg, M. L.;
Gonzalo, J. A.; Coyle, A. J.
CORPORATE SOURCE: Millennium Pharmaceuticals Inc, Cambridge, MA, 02139, USA
SOURCE: Immunological Reviews (2000), 177, 31-42
CODEN: IMRED2; ISSN: 0105-2896
PUBLISHER: Munksgaard International Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 66 refs. The understanding of the relative contribution of particular chemokines to the selective accumulation of leukocyte subsets to an organ site during an inflammatory response is made difficult by the simultaneous presence of multiple chemokines with partially overlapping functions at the inflammatory site. The study of several chemokine pathways (expression and function) during the development of a mouse model of allergic airway disease (AAD) has revealed differential expression regulation with distinct cellular sources for individual chemokines with functional bias for the recruitment/localization of regulatory and/or effector leukocyte subsets. In the present review, we propose that distinct functional groups of chemokines cooperate to generate the complete inflammatory response in the lung during AAD. We will also extend these concepts to the specific recruitment of a key cellular subset such as T helper type 2 (Th2) lymphocytes. We propose that the long term recruitment of antigen-specific Th2 cells to target organs, such as airways during chronic lung inflammation, is the result the sequential involvement of several chemotactic axes. Specifically, the CCR3/eotaxin and the **CCR4**/MDC pathway act in a coordinated cooperative manner, with the CCR3/eotaxin pathway being crit. in the acute/early stages of a response, followed by the **CCR4**/MDC pathway, which ultimately dominates in the recruitment of antigen-specific Th2 cells. Other chemokines/receptors participate in this process possibly by amplifying/priming the Th2 recruitment response.
REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L1 ANSWER 17 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:756484 CAPLUS
DOCUMENT NUMBER: 133:329593
TITLE: Low adenosine anti-sense oligonucleotide, compositions, kit and method for treatment of airway disorders associated with bronchoconstriction, lung inflammation, **allergy**(ies) and surfactant depletion
INVENTOR(S): Nyce, Jonathan W.

PATENT ASSIGNEE(S): East Carolina University, USA
SOURCE: PCT Int. Appl., 1592 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062736	A2	20001026	WO 2000-US8020	20000324
WO 2000062736	A3	20011011		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
BR 2000006019	A	20010313	BR 2000-6019	20000324
EP 1168919	A2	20020109	EP 2000-919668	20000324
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-127958P P 19990406
WO 2000-US8020 W 20000324

OTHER SOURCE(S): MARPAT 133:329593

AB An in vivo method of selectively delivering a nucleic acid to a target gene or mRNA, comprises the topical administration, e.g. to the respiratory system, of a subject of a therapeutic amt. of an oligonucleotide (oligo) that is antisense to the initiation codon region, the coding region, the 5' or 3' intron-exon junctions or regions within 2 to 10 nucleotides of the junctions of the gene or antisense to a mRNA complementary to the gene in an amt. effective to reach the target polynucleotide and reducing or inhibiting expression. In addn. a method of treating an adenosine-mediated effect comprises topically administering to a subject an antisense oligo in an amt. effective to treat the respiratory, pulmonary, or airway disease. In order to minimize triggering adenosine receptors by their metab., the administered oligos have a low content of or are essentially free of adenosine. A pharmaceutical compn. and formulations comprise the oligo antisense to an adenosine receptor, genes and mRNAs encoding them, genomic and mRNA flanking regions, intron and exon borders and all regulatory and functionally related segments of the genes and mRNAs encoding the polypeptides, their salts and mixts. Various formulations contain a requisite carrier, and optionally other additives and biol. active agents.

The low-adenosine or adenosine-free (des-A) agent for practicing the method of the invention may be prepd. by selecting a target gene(s), genomic flanking region(s), RNA(s) and/or polypeptide(s) assocd. with a disease(s) or condition(s) afflicting lung airways, obtaining the sequence of the mRNA(s) corresponding to the target gene(s) and/or genomic flanking region(s), and/or RNAs encoding the target polypeptide(s), selecting at least one segment of the mRNA which may be up to 60 % free of thymidine (T) and synthesizing one or more anti-sense oligonucleotide(s) to the mRNA

segments which are free of adenosine (A) by substituting a universal base for A when present in the oligonucleotide. The agent may be prepd. by selection of target nucleic acid sequences with GC running stretches, which have low T content, and by optionally replacing A in the antisense oligonucleotides with a "Universal or alternative base". The agent, compn. and formulations are used for prophylactic, preventive and therapeutic treatment of ailments assocd. with impaired respiration, lung **allergy**(ies) and/or inflammation and depletion lung surfactant or surfactant hypoprodn., such as pulmonary vasoconstriction, inflammation, **allergies**, allergic rhinitis, asthma, impeded respiration, lung pain, cystic fibrosis, bronchoconstriction. The present treatment is suitable for administration in combination with other treatments, e.g. before, during and after other treatments, including radiation, chemotherapy, antibody therapy and surgery, among others. Alternatively, the present agent is effectively administered prophylactically or therapeutically by itself for conditions without known therapies or as a substitute for therapies exhibiting undesirable side effects. The treatment of this invention may be administered directly into the respiratory system of a subject so that the agent has direct access to the lungs, or by other effective routes of administration, e.g. topically, transdermally, by implantation, etc., in an amt. effective to reduce or inhibit the symptoms of the ailment.

L1 ANSWER 18 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:628006 CAPLUS
DOCUMENT NUMBER: 133:217723
TITLE: Method for validating/invalidating target(s) and pathways
INVENTOR(S): Nyce, Jonathan W.
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000051621	A1	20000908	WO 2000-US5643	20000302
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
BR 2000009247	A	20011120	BR 2000-9247	20000302
EP 1165093	A1	20020102	EP 2000-913730	20000302
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002537792	T2	20021112	JP 2000-602288	20000302

PRIORITY APPLN. INFO.: US 1999-122950P P 19990305
WO 2000-US5643 W 20000302
OTHER SOURCE(S): MARPAT 133:217723
AB A method of detg. the existence of a correlation between a function of a disease or condition and a gene or mRNA encoding a target polypeptide

suspected of being assocd. with a disease or condition, comprises obtaining oligonucleotides (oligos) consisting of up to about 15 % adenosine (A), preferably having no adenosine content, and which is anti-sense to a target selected from the group consisting of target genes and their corresponding mRNAs, genomic and mRNA flanking regions selected from the group consisting of 3' and 5' intron-exon borders and the juxta-section between coding and non-coding regions, and all mRNA segments encoding polypeptides assocd. with a pre-selected disease or condition; selecting amongst the oligos one that significantly inhibits or ablates expression of the polypeptide encoded by the mRNA upon in vitro hybridization to the target mRNA; administering to a subject an amt. of the selected oligo effective for in vivo hybridization to the target mRNA; and assessing a subject's function that is assocd. with the disease or condition before and after administration of the oligo; wherein a change in the function's value greater than about 70% indicates a pos. correlation, between about 40 and about 70% a possible correlation, and below about 30% a lack of correlation. The present method preferably administers the oligos in situ where the target is located, e.g. into the subject's respiration when validating targets assocd. with malignant and other pulmonary and respiratory functions, so that the agent has direct access to the lungs. Alternatively, such desAdenosine oligos may be delivered directly to the CNS or other organs, tissues and organ systems, by known delivery formulations. This invention provides a rapid, reliable method for drug target validation/invalidation in various biol. systems that utilize proprietary low or desAdenosine antisense oligonucleotides. Using desAdenosine antisense oligonucleotides, the present method may validate/invalidate potential gene targets with a level of speed and accuracy that has heretofore been impossible using traditional techniques.

The use of antisense oligonucleotides to target adenosine receptors is described. Adenosine A1 receptor antisense oligonucleotides had bronchodilator activity in rabbits and adenosine A3 receptor antisense oligonucleotides had anti-inflammatory activity in asthmatic rabbits.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:223049 CAPLUS
DOCUMENT NUMBER: 130:251233
TITLE: Macrophage-derived chemokine (MDC), MDC analogs, MDC inhibitor substances,and their therapeutic applications
INVENTOR(S): Gray, Patrick W.; Chantry, David H.; Deeley, Michael C.; Raport, Carol J.; Godiska, Ronald
PATENT ASSIGNEE(S): Icos Corporation, USA
SOURCE: PCT Int. Appl., 159 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9915666	A2	19990401	WO 1998-US20270	19980928
WO 9915666	A3	19990916		

SOURCE: International Archives of Allergy and Immunology (2001), 124(4), 423-431
CODEN: IAAIEG; ISSN: 1018-2438
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Chemokines are a group of cytokines that are responsible for the influx of blood cells, including T and B lymphocytes, monocytes, neutrophils, eosinophils and basophils, in allergic and other inflammatory conditions. They function as G protein-coupled chemotactic factors which also activate the cells with which they interact. Certain chemokines function within the afferent arm of the immune system, in which antigen is processed and antibody formation initiated, and others are active within the effector pathways of cellular immunity and late-phase allergic reactions. Th2 lymphocytes, which are crit. for **allergy**, employ the CC chemokine receptors CCR4 and CCR8 with the ligands thymus- and activation-regulated chemokine (TARC), macrophage-derived chemokine (**MDC**) and I-309, resp. The chemokine receptor CCR3 and ligands monocyte chemoattractant protein (MCP)-3, MCP-4, regulated upon activation normal T cell expressed and secreted (RANTES) and eotaxins I and II are of particular relevance for the recruitment and activation of eosinophils. Th1 reactions depend upon interferon .gamma.-induced CXC chemokines interferon-inducible protein (IP)-10, interferon-inducible T cell-.alpha. chemoattractant (iTAC) and monokine induced by interferon-.gamma. (MiG), which bind to chemokine receptor CXCR3.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:296102 CAPLUS
DOCUMENT NUMBER: 135:75647
TITLE: Stem cell factor and IgE-stimulated murine mast cells produce chemokines (CCL2, CCL17, CCL22) and express chemokine receptors
AUTHOR(S): Oliveira, S. H. P.; Lukacs, N. W.
CORPORATE SOURCE: University of Michigan Medical School - Department of Pathology, Ann Arbor, MI, 48109-0602, USA
SOURCE: Inflammation Research (2001), 50(3), 168-174
CODEN: INREFB; ISSN: 1023-3830
PUBLISHER: Birkhaeuser Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Objective and design: In the present study we investigated the effect of SCF and/or IgE on histamine, TNF-.alpha. and chemokines released from bone marrow-derived mast cells (BMMC) as well as chemokine receptor expression.
Material and methods: BMMC were derived from femoral bone marrow of CBA/J mice. The purity of BMMC was >98% after 3 wk. BMMC (2.5 .times. 106 cells/well) were incubated in the presence or absence of either SCF, IgE plus DNP or a combination of SCF and IgE for 6 and 18 h. Cell-free supernatants were recovered to measure CC chemokines, TNF-.alpha. and histamine release utilizing ELISA assays. CC chemokine family receptors were detected by RT-PCR anal., and confirmed using functional chemotactic

assays. Results: Histamine levels were comparable between SCF and IgE stimulated cells, whereas TNF-.alpha. prodn. was significantly greater after IgE compared to SCF stimulation. SCF and/or IgE-stimulated BMMC released CC chemokines, CCL22 (**MDC**), CCL17 (TARC) and CCL2 (MCP-1). Increased mRNA expression of CCR1, CCR2, CCR3, and CCR5 was detected in SCF and IgE-stimulated BMMCs. Functional chemotactic assays confirmed the expression data. Conclusion: SCF and IgE can up-regulate the expression of chemokines and chemokine receptors on mast cells. Thus, SCF may play a significant role in their activation and inflammation during allergic responses.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:903436 CAPLUS

DOCUMENT NUMBER: 135:120758

TITLE: Non-redundant functional groups of chemokines operate in a coordinate manner during the inflammatory response in the lung

AUTHOR(S): Gutierrez-Ramos, J. -C.; Lloyd, C.; Kapsenberg, M. L.; Gonzalo, J. A.; Coyle, A. J.

CORPORATE SOURCE: Millennium Pharmaceuticals Inc, Cambridge, MA, 02139, USA

SOURCE: Immunological Reviews (2000), 177, 31-42
CODEN: IMRED2; ISSN: 0105-2896

PUBLISHER: Munksgaard International Publishers Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 66 refs. The understanding of the relative contribution of particular chemokines to the selective accumulation of leukocyte subsets to an organ site during an inflammatory response is made difficult by the simultaneous presence of multiple chemokines with partially overlapping functions at the inflammatory site. The study of several chemokine pathways (expression and function) during the development of a mouse model of allergic airway disease (AAD) has revealed differential expression regulation with distinct cellular sources for individual chemokines with functional bias for the recruitment/localization of regulatory and/or effector leukocyte subsets. In the present review, we propose that distinct functional groups of chemokines cooperate to generate the complete inflammatory response in the lung during AAD. We will also extend these concepts to the specific recruitment of a key cellular subset such as T helper type 2 (Th2) lymphocytes. We propose that the long term recruitment of antigen-specific Th2 cells to target organs, such as airways during chronic lung inflammation, is the result the sequential involvement of several chemotactic axes. Specifically, the CCR3/eotaxin and the CCR4/**MDC** pathway act in a coordinated cooperative manner, with the CCR3/eotaxin pathway being crit. in the acute/early stages of a response, followed by the CCR4/**MDC** pathway, which ultimately dominates in the recruitment of antigen-specific Th2 cells. Other chemokines/receptors participate in this process possibly by amplifying/priming the Th2 recruitment response.

REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:68155 CAPLUS
DOCUMENT NUMBER: 132:106969
TITLE: Chemokines as adjuvants of immune response
INVENTOR(S): Caux, Christophe; Vanbervliet, Beatrice; Lebecque, Serge; Vicari, Alain; Dieu, Marie-Caroline
PATENT ASSIGNEE(S): Schering-Plough, Fr.
SOURCE: Eur. Pat. Appl., 16 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 974357	A1	20000126	EP 1998-401799	19980716
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
WO 2000003728	A1	20000127	WO 1999-US14148	19990715
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MD, MG, MK, MN, MX, NO, NZ, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9949591	A1	20000207	AU 1999-49591	19990715
US 2002034494	A1	20020321	US 2001-768917	20010124
WO 2002058723	A2	20020801	WO 2002-US1849	20020122
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VN, YU, ZA, ZM, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
EP 1998-401799 A 19980716
WO 1999-US14148 W 19990715
US 2001-768917 A 20010124

AB Dendritic cells play a crit. role in antigen-specific immune responses. Materials and methods are provided for treating disease states, including cancer and autoimmune disease, by facilitating or inhibiting the migration or activation of antigen-presenting dendritic cells. In particular, chemokines are used to initiate, amplify or modulate an immune response. In one embodiment, chemokines are used to attract dendritic cells to the site of antigen delivery. An increase no. of dendritic at the site of antigen delivery means more antigen uptake and a modified immune response.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:796268 CAPLUS